Burns to the Head, Face and Neck: PART I

41

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Facial burns vary from relatively minor insults to severe debilitating injuries. Over 50% of burn injuries involve the head and neck region and can be caused by flame, electrical current, steam, hot materials/liquids and chemicals. In addition to the obvious disfigurement that arises from these, burns can also have a major impact on important everyday functions such as seeing, hearing and breathing. Moreover, significant facial burns can be detrimental to mental wellbeing and personal self-esteem. Treatment of facial burns is more demanding than treatment of burns elsewhere, not only because of the location of vital organs, but because the face is highly vascular. This high vascularisation increases the healing potential of facial burns and therefore, justifies a more conservative approach to treatment. However this may require intensive and daily treatment. Because there remains uncertainty about which treatment is the most effective for facial burns, large variations in practice occur. All facial burns should therefore be discussed with your local burns unit, or specialist.

Burns can be classified into six main types, based on the mechanism of injury

1. Scalds (liquids, grease or steam). Liquid scalds can be further divided into spill and immersion scalds.
2. Contact burns—cookers, hot metal
3. Fire (flash and flame)
4. Chemical—acid and alkali
5. Electrical
6. Radiation—severe sunburn and exposure to nuclear blasts (historically in Japan, towards the end of the second world war)

These mechanisms can be useful predictors of outcome — for example, flame and electrical burns often require admission, where as burns from hot surfaces or sun exposure can usually be managed as outpatients.

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M. Perry (ed.), *Diseases and Injuries to the Head, Face and Neck*,
https://doi.org/10.1007/978-3-030-53099-0_41

41.1 Applied Anatomy

Detailed anatomy of the skin is (perhaps not surprisingly), discussed in the chapter on the Skin. The skin has many important functions including (1) mechanical protection, (2) maintaining body temperature, (3) preventing infection, (4) gathering sensory information, (5) hormone production and (6) regulating the immune system. All of these can be compromised to varying extents by a burn. Functionally, skin consists of three main layers, (1) the epidermis (stratified squamous epithelium), (2) the dermis (connective tissue) and (3) subcutaneous tissue (fat). Scattered throughout the dermis and epidermis are numerous epidermal appendages, notably sweat glands, sebaceous glands and hairs. It is from these appendages that the epidermis can regenerate following partial thickness burns.

41.1.1 Epidermis

The thickness of the epidermis varies according to its site. It is the thinnest in the eyelids, making them highly sensitive to any type of burn. Epidermis is composed of 5 layers (Basal, Spinosum, Granulosum, Lucidum and Corneum). The deepest layer, the stratum basale, contains the cells that divide and push already formed cells into the more superficial layers, amassing keratin and flattening as they go. The top layer of the epidermis, the stratum corneum, is made of dead, flat cells that shed about every 2 weeks. The skin acts as a barrier to impede the entrance of microorganisms and toxic substances, whilst allowing the body to retain water and electrolytes. Both of these functions are severely disrupted following burns. This can have a major impact on morbidity and management if extensive.

41.1.2 Dermis

The dermis varies in thickness from 0.3 mm on the eyelid to 3.0 mm on the back. It consists of collagen, elastic tissue and reticular fibres. There is an upper papillary layer and a lower reticular layer. The dermis contains many specialised cells and structures. These include (1) hair follicles with erector pili muscles, (2) sebaceous (oil), eccrine (sweat) and apocrine (scent) glands, (3) blood vessels and nerves (4) Langerhan cells (5) Meissner's, Merkel and Vater-Pacini corpuscles (touch and pressure sensors). Cells in the glands are also important in regeneration following partial thickness skin loss.

41.1.3 Subcutaneous Tissue

The subcutaneous tissue is a layer of fat and connective tissue, containing larger blood vessels and nerves. This layer is important is the regulation of temperature. The size of this layer varies throughout the body and from person to person.

41.2 Pathophysiology

The effects of burn injury extend far beyond the skin. It is truly a systemic disease. Locally, the severity of an injury is related to the rate at which heat is transferred to the skin. This depends on the temperature of the burning agent, its specific heat capacity (the quantity of heat energy the material contains), duration of contact, transfer coefficient and the conductivity of the local tissues. Water has a high specific heat capacity—it takes a lot of heat to melt ice and to boil water. It is actually one of the highest specific heat capacities of many of the gases, metals and solids that are likely to result in burn injuries. This is why scalds and steam injuries can be so severe.

Heat can be transferred by conduction, convection and radiation. Conduction occurs when a hot solid object comes in direct contact with the skin. Convection occurs when particles with a lot of heat energy in a liquid or gas move and take the place of particles with less heat energy. It is the mechanism whereby heat passes from a hot liquid or gas. Hot water, steam and very hot air burn by convection (and some conduction). Radiation (infrared) is a type of electromagnetic radiation. There does not need to be any contact with the source (as seen in sunburn).

Within the skin itself, heat transfer depends on the amount of heat applied, the area over which it is transferred, its water content, natural oils and secretions and the thickness of its insulating stratum corneum (keratin layer). Local blood flow also affects heat transfer and distribution. Skin is a relatively poor conductor of heat and so provides an insulating barrier for the inner structures. This ability can vary depending on its thickness. The rarity of full-thickness burns on the palms and soles of the feet can be attributed to their thick epithelial cover. In most sites on the body skin can tolerate temperatures as high as 44 °C (111 °F) for a relatively long time (6 h) before irreversible injury occurs. However, temperatures greater than this cause a rapid increase in tissue destruction. When hot water is splashed on a person, it usually flows downwards unless absorbed or deflected by clothing. Viscous oils and greases on the other hand usually adhere to the skin, prolonging the duration of contact and the extent of the burn. In immersion scalds, the duration of contact between the hot liquid and the skin can be relatively high, resulting in very severe burns. Immersion burns also tend to cover a larger percentage of the total body surface area (TBSA), and thus have high morbidity and mortality.

When skin is burned, the damaged vessels increase their permeability and extravasation of fluids and electrolytes occurs. If extensive, this can quickly result in hypovolemia with decreased tissue perfusion and oxygen delivery. Increased evaporative water loss and exposure of the deeper tissues can also result in hypothermia. When a burn exceeds 30% of TBSA, cytokines and other mediators are released into the systemic circulation, causing a systemic inflammatory response. Release of catecholamines, vasopressin, and angiotensin causes peripheral and splanchnic vasoconstriction, which can further compromise organ perfusion. The term “burn shock” is often used to indicate the massive physiologic response that ensues. This can affect nearly every organ system. The extent of this shock is proportional to the size of the burn area. A decrease in pulmonary function can also occur, even without

any inhalation injury. Bronchoconstriction is reported to develop following the release of histamine, serotonin and thromboxane A2. In severe burns adult respiratory distress syndrome can thus occur. Myocardial contractility may also be reduced, possibly due to release of tumour necrosis factor. All these changes may be complicated by haemolysis of red cells. In severe burns, the RBC mass may fall up to 15%. Microangiopathic haemolytic anaemia may persist for several weeks.

Burns (with the exception of alkalis) coagulate tissue proteins and result in necrosis of tissue. This is called coagulative necrosis. The main difference between thermal and chemical burns is the period of contact. Chemical burns will continue to destroy tissues until the substance is neutralised or removed. Alkali burns tend to penetrate more deeply than acids by a process of 'saponification' and by liquefactive necrosis. Electrical burns are more complex. Cell damage is caused by two separate processes (1) direct thermal burn (current-generated heating) and (2) electroporation (the electrical current creates large 'holes' in the cell membrane, which allows ions, metabolites, and even DNA to escape). Most fire-related deaths result not from the burn itself but from inhalation of the toxic products of combustion. This depends on the material burnt and the concentration and the solubility of the gases produced.

Following extensive burns, the immune response is also diminished, making the patient very susceptible to serious infections. Major burns are defined as a burn greater than 25% TBSA. Locally, damaged cells enable the invasion of microorganisms. Damaged Langerhans cells, which mediate local immune responses, are unable to cope with the biological insult. Much of the morbidity and mortality that comes from severe burns is therefore attributable to (1) the systemic inflammatory response and (2) overwhelming sepsis. A hypermetabolic response can last for many months and is associated with impaired wound healing, increased risk of infection, loss of body mass and impaired recovery. Added to this are the severe psychological challenges the patient experiences when facial burns result in significant disfigurement.

41.3 Principles of Management for all Burns

As a general rule, always follow local protocols if these are available. If the history suggests a possibility of coexisting injuries (e.g. blast injuries or a fire in a collapsing building), the initial approach is the same as with any other trauma victim (See chapter on the injured patient). Key questions should include (1) The time of injury (for fluid resuscitation), (2) Details of the environment (whether the patients was in a closed or open space) (3) The mechanism of escape from the scene of fire. This may give some idea about any associated injuries (did they jump from a window?) (4) Any pre existing illnesses—Diabetes, hypertension, cardiopulmonary and renal problems are of particular relevance in burns management, (5) Drug history, (6) Allergies and (7) Tetanus status. Then carry out a rapid primary survey, followed by the detailed secondary survey. However, when the mechanism of injury is solely

that of a burn, the Burn Primary Survey may be used. This is often abbreviated to ABCDEF.

1. Airway
2. Breathing
3. Circulation
4. Disability
5. Expose (and assess burn depth and TBSA)
6. Fluids (Parkland Formula)

In all burns patients there should always be a high index of suspicion for inhalation injury and the possible need for early intubation, tracheostomy and escharotomies. Airway compromise as a result of thermal injury or oedema can arise quickly and make intubation difficult. If in doubt, intubate early or seek senior help.

41.3.1 Stop the Burning Process as Soon as Possible

Clothing and viscous gels can retain heat and should be removed as soon as possible. Any adherent material should be left on, such as melted nylon clothing, plastic or tar. However, corrosive agents will continue to cause necrosis until they are removed. Alkalis penetrate deeper than acids and the resultant burns tend to be more severe. Cement burns are an example of an alkali burn. Patients may not present until the burn is full thickness. Initial management involves careful removal of any chemical-soaked clothing or chemical powder. The majority of chemicals should be diluted with copious irrigation (using water, rather than trying to neutralise the chemical). Litmus paper can be used to check the effectiveness of the irrigation. Some chemical burns can be very problematic. For example, burns caused by sodium, potassium or lithium will spontaneously ignite when exposed to water. These elements should therefore be buffered with diphoterine to neutralise their effect. Oxalic and hydrofluoric acids (widely used for glass etching and in the manufacture of circuit boards) can result in severe hypocalcaemia and hypomagnesaemia. They require treatment with topical and IM 10% calcium gluconate. Chromic acid and dichromate salt burns should be rinsed with dilute sodium hyposulphite. In all burns if there is ocular or eyelid involvement an ophthalmology review is essential.

41.3.2 Cool the Burn

First-aid measures include irrigation with tepid water (15 °C) for 20 min or the application of a saline-soaked cloth to decrease heat damage. Dry chemical powder burns need careful inspection and removal of any dry particles before applying water. Then dilute the remaining chemical with copious irrigation. Attempting to

chemically neutralise an acid or alkali can produce an exothermic reaction, resulting in further burning.

41.3.3 Calculate the Depth and Area of Burn

Ideally fluids should have been started during the initial survey. It is during "E", when the patient is exposed, that an accurate assessment of the depth and area of the burn is then undertaken. This allows fluid resuscitation to be calculated. The Parkland formula is a universally known guide for this. Resuscitation should be discussed with the receiving burns unit as soon as possible.

41.3.4 Analgesia

Exposed nerve endings are extremely painful. Paradoxically the deeper the burn, the less pain a patient will experience. Simply cooling and covering the burn will improve pain. Opioids are often the first line of analgesia but careful titration is required especially if there is any coexisting head injury. This should be given as an initial intravenous bolus of morphine (proportional to body weight) and titrated to the response. The patient should be reviewed every 30 min and further doses given as required. In smaller burns NSAIDs taken orally should suffice.

41.3.5 Prevention of Infection

The most common organisms are *Staphylococcus aureus* and *Pseudomonas aeruginosa*. Severe microbial colonisation can result in delayed wound healing, and increased length of hospitalisation. Wounds must therefore be kept as clean as possible using topical antimicrobial agents. However, as a general rule, systemic antibiotics are not required for minor burns in adults. With paediatric burns, some units recommend a 5-day course of flucloxacillin to prevent toxic shock syndrome. In patients with major burn injuries, infection remains the major cause of death. This includes systemic candidiasis. The administration of antibiotics should therefore only be considered following discussion with the receiving burns unit.

41.3.6 Nutritional Support

Burns initiate a physiological cascade that results in a hypermetabolic state. This is characterised by a dramatic increase in resting energy expenditure Nutritional support is therefore essential, preferably via the enteral route, to reduce intestinal villous atrophy. Decreased bowel mucosal integrity, capillary leakage and decreased mesenteric blood flow have been reported to allow the passage of luminal bacterial into the portal circulation. In severe cases this can result in systemic sepsis and

multi organ failure. Prevention requires not only fluid resuscitation (to ensure mesenteric blood flow) but also enteral nutrition containing glutamine (which preserves mucosal integrity).

41.3.7 Dressings

A dressing should cover the burn area and keep the patient warm. Clingfilm is a useful temporary dressing, which allows assessment of the burn with minimal distress to the patient. Clingfilm is reported to be sterile as it comes off the roll and should be laid over the burn rather than wrapped around it. As the burn swells tight dressings can lead to constriction. A blanket placed on the patient will keep the patient warm. Avoid the application of flamazine (silver sulphadiazine) for the first 48–72 h. Flamazine is a useful topical antimicrobial but it makes burns look deeper than they actually are. If you apply flamazine prior to transfer, you will be the only person to have seen the true extent of the burn and further assessment will be impossible. Flamazine should not be applied to the face, as the silver can disturb pigmentation.

41.3.8 Other Measures

A urinary catheter must be inserted to record urinary output. Fluid resuscitation should be altered according to the hourly output. With large burns (>20%), a nasogastric tube should also be placed to facilitate gastric decompression. Without decompression, gastric ileus can develop resulting in nausea, abdominal distension and vomiting. A nasogastric tube can be also used to administer antacids and provide enteral feeding. Burns are hugely catabolic and a burn >50% total body surface area (TBSA) increases the basal metabolic rate by approximately 200%. A flow sheet, outlining the patient's management should also be initiated as soon as the patient attends the emergency department. This should accompany the patient to the burns unit if transfer is required. Clinical photographs are a useful record of events. The area and depth of burn should also be recorded on an appropriate chart (e.g. Lund and Browder).

41.4 Burn Primary Survey

41.4.1 Airway Considerations

Rapid assessment of the airway needs to quickly establish whether it is compromised or may become compromised by the burn. If mechanical trauma is suspected, the c-spine should be simultaneously immobilised until a cervical spine injury has been ruled out. In most cases the larynx protects the subglottic airway from direct thermal injury. However, the supraglottic passage is quite vulnerable to heat

exposure and may quickly obstruct as swelling progresses. Direct thermal injury to the upper airway and lungs can rapidly produce upper airway oedema and obstruction. This is especially the case if steam has been inhaled—steam has a high latent heat of evaporation so releases a lot of thermal energy when it cools. Steam inhalation therefore has a very high mortality rate. The skin and soft tissues of the neck itself may also swell or contract with eschar, compromising the airway. Therefore consider intubation early and involve an experienced anaesthetist in the assessment of the patient.

41.4.2 Breathing Considerations

Breathing problems are mostly caused by thermal injuries and irritants passing below the vocal cords, but they can occur following secondary bronchoconstriction in response to inflammatory mediators. Suspect inhalation injury in

1. All facial burns
2. Singeing of eyebrows and nasal vibrissae
3. Carbonaceous sputum
4. Carbon deposits and swelling mouth and throat
5. A history of confinement
6. Confusion
7. Following explosions with burns to head and torso
8. A carboxyhaemoglobin greater than 10%

Symptoms suggestive of inhalation injury may include

1. Lacrimation
2. Severe brassy cough
3. Hoarseness
4. Shortness of breath
5. Anxiety
6. Wheezing

All patients should receive 100% oxygen through a humidified non-rebreathing mask immediately on presentation. Rapidly assess (and regularly reassess) for signs of respiratory distress. Patients with inhalation injuries are at risk of

1. Inhalation of toxic fumes. This can lead to chemical trachebronchitis, oedema, and pneumonia
2. Carbon monoxide poisoning
3. Secondary bronchoconstriction

Furthermore, if the chest has been burnt there is also a risk of eschar contraction compromising chest movement. Blast injuries can result in “blast lung”, with alveolar contusion and rapid onset of hypoxia. Clinical manifestations of smoke-inhalation injury are subtle in the early stages and may not become obvious for 24 h.

The decision to intubate is often a clinical one, rather than based on the findings of a chest X-ray or blood gas results. A high index of suspicion must therefore always be maintained and an anaesthetic opinion and fibre optic bronchoscopy obtained if there are any concerns. Arterial blood gases should be determined as a base line prior to intubation.

Always assume carbon monoxide (CO) exposure in any patient involved in a burn in a closed environment. Disorientation, drowsiness and coma indicate significant exposure to carbon monoxide (in the absence of other injuries). Carbon monoxide binds to deoxyhaemoglobin with 240 times the affinity for oxygen, shifting the oxygen dissociation curve markedly. In addition, it attaches to intracellular proteins and inhibits the cytochrome oxidase pathway involved in respiration. Both these actions result in profound intra and extra cellular hypoxia. Unfortunately, pulse oximetry is often normal as oxyhaemoglobin and carboxyhaemoglobin are indistinguishable to the probe. CO levels of <20% may not always be symptomatic. Symptoms may initially be subtle and include headache and nausea (20–30%), confusion (30–40%), coma (40–60%) and eventually death (>60%). Blood gases should therefore be taken. These will reveal a metabolic acidosis and raised carboxyhaemoglobin levels. Carboxyhaemoglobin levels from blood can sometimes help determine the full extent of exposure at the time of injury. Nomogram charts are now available to extrapolate the level obtained and use this to calculate the original levels of CO. All patients suspected of exposure to CO should be administered high-flow 100% oxygen via a reservoir mask.

41.4.3 Circulatory Considerations

With increasing TBSA involvement, patients can quickly loose fluids. Burns >10% TBSA in children and > 15% TBSA in adults require IV fluid resuscitation. Intravenous access should therefore be secured, preferably through unburnt skin. Bloods samples should be taken and sent for full blood count, urea and electrolytes, clotting, amylase and a group and save. Carefully assess the peripheral circulation for both signs of hypovolaemia and impaired limb perfusion. It is unusual for a burns patient to be hypovolaemic immediately after injury so if this is noted, consider other injuries and cardiogenic shock. Remember also that a circumferential eschar can impair the circulation to a limb.

41.4.4 Neurological Considerations

All patients should be assessed using the Glascow Comma Scale. Hypoxia, hypovolaemia or CO poisoning can all cause diagnostic confusion.

41.4.5 Exposure with Environmental Control

The whole of the patient should be exposed to assess for burns or concomitant injuries (particularly the back). Patients should be covered and warmed as soon as possible. Hypothermia will lead to hypoperfusion and deepening of the burn wound.

41.4.6 Fluids

Burns in children >10% TBSA and > 15% TBSA in adults require IV fluid resuscitation. A urinary catheter should be inserted in any patient with a burn >20% TBSA to monitor urinary output.

41.4.7 The Secondary Survey

Once the primary survey has been performed, the secondary survey should begin. This is a head to toe examination for any other injuries that may have been overlooked in the primary survey. It includes investigations including an ECG, chest x-ray and other x-rays as appropriate. All patients with facial burns should be suspected of having a corneal injury. The secondary survey should therefore include an early ophthalmic examination using fluorescein dye—eyelid swelling can develop rapidly. If there is any corneal involvement then the patient should be referred for an ophthalmic opinion and given chloramphenicol eye ointment.

41.5 Assessing a Burn

Following the physiological assessment of the patient and correction of any deficits, attention can be turned towards the burn. Several key characteristics need to be determined (1) the extent (burn area) (2) depth and (3) involvement of important structures (such as the eyelids, mouth or hands).

41.5.1 Assessment of Burn Area

Record the patient's weight in kilograms. This is important for calculating fluid requirements and in assessing nutritional requirements later on. Accurate assessment of the extent of a burn is important in its ongoing management, prognosis and calculation of fluid requirements. This is often done poorly as it is often difficult to assess TBSA accurately. The palmar surface area is often used as a guide. The surface of a patient's palm (from finger tips to wrist crease) equates to approximately 0.8% of body surface area. This is useful for smaller or irregular patches of burns. When calculating the burn area simple erythema is not included. For more extensive burn, the 'rule of nines' may be used to approximate the extent of the burn. In adults, the body can be divided into anatomical regions, which represent 9% or multiples of 9% of the total body surface. Hence head and neck = 9%, each upper limb = 9%, anterior torso = 18%, posterior torso = 18%, each lower limb = 18%, perineum = 1%. This proportion is not applicable in children, as they have a larger surface area: body weight ratio and the head accounts for more of this.

41.5.1.1 Lund and Browder Charts

Alternative to these methods, special charts are now often available for estimation of burn area at all ages. These are more accurate than the 'rule of nines' and should be available in every Emergency department. They also help in the graphic representation of the area and the depth of a burn and can be used as a record following transfer.

41.5.1.2 Burn Zones

During the first day after a burn injury, three concentric zones develop in the wound

1. Zone of central coagulative necrosis—this consists of non-viable tissue and is the site of the most contact with the heat source. This zone consists of dead or dying cells (coagulation necrosis) and an absent blood flow. It usually appears white or charred.
2. Zone of intermediate stasis—this tissue is initially viable, but has a precarious blood supply. It initially appears red and may blanch on pressure, indicating some residual circulation is present. Petechial haemorrhages may be present. One of the aims of burns management is to prevent this zone progressing to necrosis. If this is not achieved this zone will become part of the central zone of necrosis and the burn will extend. Despite fluid management in many cases the circulation through the superficial vessels often becomes compromised and ceases. This is believed to occur as a result of progressive dermal ischaemia and the local effects of prostaglandins, histamine and bradykinin, which have been linked to progressive vascular occlusion. Progressive tissue oedema also compromises the microcirculation. By the third day, the tissues become white as the superficial dermis becomes avascular and necrotic.
3. Outer zone of hyperaemia—this is the outermost area of a burn, seen as an inflammatory response. It appears red and blanches/reperfuses briskly on pressure, indicating that it has an intact and hyperaemic circulation. In burns >25% Total Body Surface Area (TBSA), the whole of the body may be included in this zone. By the fourth day, this has a deeper red appearance. Healing has usually occurred by the seventh day.

41.5.2 Depth of Burn

This is directly related to the temperature applied and duration of burn, as well as the thickness of the skin. Initial assessments of burn depth may not be accurate as a superficial burn may progress to a deeper burn following infection or inadequate resuscitation. Repeated examinations over a few days are therefore necessary. The British Burn Association (BBA) recommends the following classification.

41.5.2.1 Superficial

This is erythema only and should not be included in any of the assessments of burn area. It involves minimal tissue damage and is confined to the epidermis. Common

causes include sunburn, scalds or flash flame. The skin appears dry and without blisters, with a pink colour that is usually painful. Healing occurs over 5–10 days. Usually, no permanent scar occurs, but the tissue may discolour.

41.5.2.2 Superficial Dermal

These burns can be caused by contact with hot liquids or solids, flash flame or chemicals. They involve the epidermis and most superficial dermis only. There is typically erythema and moist blistering, which is very painful. Although a variable depth of skin is usually lost, most partial-thickness burns spontaneously re-epithelialise without scarring if treated appropriately. This takes about a week.

41.5.2.3 Deep Dermal

These extends deeper into the dermis but skin adnexal structures are still undamaged, allowing spontaneous healing. The wound feels dry to touch and may take up to 3 weeks to heal if uncomplicated by infection. A deeper burn may take more than 30 days to heal and can convert to a full-thickness injury if it becomes infected. To improve scarring surgical management is often required.

41.5.2.4 Full Thickness

These burns can be caused by contact with hot liquids/solids, flames, chemicals or electricity. All the dermal elements are destroyed. The skin appears translucent, mottled or waxy white. If there is significant leak of red blood cells from damaged vessels, it may have a deep red appearance. Later, it turns brown or black following eschar formation. These areas are usually painless and insensate because of the loss of sensory nerve endings. There may be involvement of underlying subcutaneous tissue, muscle, tendons or bones with thrombosed vessels. Most of these wounds do not heal spontaneously unless they are very small and the resulting scars may be quite disfiguring. Surgical intervention is usually required.

41.5.3 Burns Involving Key Structures

Burns involving the eyelids, nose, mouth, ear, and scalp cause significant functional and cosmetic problems. These patients also need a great deal of psychosocial rehabilitation in later life. All patients with facial burns should be suspected of having a corneal injury.

41.5.3.1 Burns to the Ears and Nose

Burns of the ear invariably demonstrate a mixture of the three degrees of burn. They are characterised by central coagulation, with peripheral areas of stasis and hyperaemia. Because the cartilage is avascular, nutritional disruption results in tissue loss and deformity. Chondritis is common resulting in resorption of cartilage and ultimately deformity of the ear. Management is often conservative in the first instance. The wound is thoroughly cleaned and sterile paraffin ointment or gauze is often applied. Mafenide (Sulfamylon) cream is a good topical antibiotic suitable for the ear because of its ability to penetrate any eschar and the cartilage.

It has a broad antibacterial spectrum with good bacteriostatic effect and relatively low toxicity. Full-thickness which result in perichondritis may need excision after 3–4 weeks.

41.5.3.2 Burns to the Eyes

Patients who are conscious at the time of burn often tightly close their eyes, thereby protecting the eyelids and cornea. However, unconscious patients, such as epileptics, head injured etc., are often at risk of corneal damage due to the thin skin of the eyelids. During initial assessment it is important not miss the opportunity of a careful ophthalmic examination using fluorescein dye. Eyelid swellings develop rapidly and an early opportunity may be quickly lost. If the patient has a corneal ulcer, then they should be referred for an ophthalmic opinion and given chloramphenicol eye ointment.

41.5.3.3 Burns to the Mouth

These are particularly difficult to manage. Fluid and nutritional intake may be significantly impaired and circumoral burns can contact tightly, severely narrowing the oral aperture. If the burn is secondary to liquids, consider also the possibility of aspiration and ingestion.

41.5.3.4 Burns to the Scalp

These often result in tissue loss, exposed bone and the need for reconstruction once the tissues have recovered. Hair loss is also common.

41.5.4 Fluid Resuscitation and Requirements

Fluid losses from burns must be replaced precisely in order to optimise the patient's recovery. The greatest loss occurs within the first 24 h as the tissues swell and water and electrolytes are lost from the damaged skin. Currently there is no perfect formula for fluid resuscitation. Several formulas exist but these are all just guides and the fluids given must be tailored according to the patient's physiological response. In the UK most units use the Parkland formula. In this regime the amount of fluid to be infused is calculated based on the time of injury, not the time of arrival in the emergency department (which may be hours later). If the arrival of the patient is delayed, then it is important to 'catch up' the deficit.

41.5.4.1 Parkland Formula

4 ml × weight (kg) × %TBSA = number of mls of Hartmann's solution to be given in 24 h. Half of this volume should be given during the first 8 h and the remainder over the next 16 h. In addition to this, children should receive maintenance fluids at an hourly rate of

- 4 ml/kg for the first 10 kg *plus.*
- 2 ml/kg for the second 10 kg *plus.*
- 1 ml/kg for every kg above 20 kg.

The goal of the fluid resuscitation is to obtain a urine output in the range of 0.5–1 ml/kg/h adults and 1–1.5 ml/kg/h for children. A urinary catheter should therefore be inserted to monitor the urinary output in any burn over 20% TBSA. Adjustments in the resuscitation fluid are then made based on patient's response. A fluid bolus is recommended only in the presence of marked hypotension, not a low urine output.

41.6 Management of Minor Burns

Burns suitable for outpatient management include

- Partial thickness burns in adults <10% BSA
- Partial thickness burns in children <5% BSA
- Full thickness burns <1% BSA
- No co-morbidities

Fresh wounds are usually sterile and every attempt should be made to keep them so. The wound should be cleaned with soap and water or dilute chlorhexidine. The management of blisters is controversial. Many units advocate leaving smaller blisters, but de-roofing larger ones which compromise function (aseptic technique). After the wound has been cleaned a variety of dressings can be applied. There is little evidence to support any particular dressing.

For facial burns, dressings can be difficult to apply and should be discussed with a specialist unit. Superficial burns are best managed open with dilute chlorhexidine washes twice daily and paraffin gauze applied every 4 hours to minimise crusting. For deeper burns, a simple sterile gauze dressing impregnated with Vaseline (jelonet), covered with a gauze pad, cotton wool and a crepe dressing will provide a sterile and absorbent cover with minimal slippage. Thereafter, the dressing should be inspected at 24 h to ensure there is no leakage and the wound itself re-inspected at 48 h. At this stage, the depth of the burn should be obvious and agents such as flamazine can be used (except on the face). Dressing changes can then be performed every 3–5 days. If a burn has not healed by 2–3 weeks then it should be seen by a specialist burn surgeon. Aggressive exercises and stretching should be commenced in partial-thickness orofacial burns soon after hospital admission. This has been shown to prevent orofacial contractures.

The National Burn Care Review has published guidelines on referral for burns patients to specialist units. These include:

- Extremes of age (<5 years or > 60 years)
- Site of injury (hands, face, perineum, feet, flexures, circumferential burns
- Inhalational injury
- Chemical, steam, radiation, electrical, HF acid, non accidental burns
- Large area dermal or full thickness burn (>5% in a child or > 10% in an adult)
- Coexisting or concomitant injury

41.6.1 Electrical Burns

Electrical burns are uncommon in the head and neck, and if they occur are usually fatal. Involvement of the face in survivors can be very extensive and hugely disfiguring. The current travels through the body from the point of contact, creating entry and exit wounds. All the tissues between these two points can be damaged to varying degrees. The greatest generation of heat occurs when electrical energy passes along a tissue of high resistance, such as bone. Low voltage burns from domestic current often result in small, deep entrance and exit wounds. Cardiac dysrhythmias can occur and an ECG should be obtained in all patients. If the ECG is normal and there is no history of loss of consciousness then cardiac monitoring is not required. High voltage burns cause extensive tissue damage. Ventricular fibrillation is a common cause of immediate death. Survivors must be monitored for compartment syndrome, haemochromogenuria and cardiac dysrhythmias. Muscle damage leading to rhabdomyolysis can lead to renal failure. Patients can also have associated fractures, corneal injury, and tympanic perforation. Flash burns are caused by arcing from a high tension voltage source. These cause superficial flash burns due to their heat but no current passes through the patient.